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Methylene blue and peritoneal dialysis

To the Editor: I have read with interest the case presented by Wu *et al.*[1] regarding urinary bladder perforation after implantation of a peritoneal dialysis (PD) catheter. The colored picture showing methylene blue in the effluent dialysate in the PD bag is quite impressive for the general practitioner, but nephrologists involved in the field of PD may express several objections regarding this approach.

There are several reports that methylene blue may be an irritant to the peritoneum and cause chemical peritonitis, and thus, it is not advisable to use it for diagnostic reasons in PD.[2,3] The unexpected glycosuria and the abdominal computed tomography imaging should be sufficient to establish the correct diagnosis, as in other cases.[4]

The patient of Wu *et al.*[1] was transferred to maintenance hemodialysis. However, I would not consider the use of methylene blue in PD for diagnostic reasons as a wise approach, as the main focus of PD remains to preserve the peritoneum for as long as possible.

1. Wu HH, Li SY, Yang WC. Make your diagnosis: adynamic ileus after insertion of peritoneal dialysis catheter. *Kidney Int* 2010; **78**: 525–526.
2. Macia M, Gallego E, Garcia-Cobaleda I *et al.* Methylene blue as a cause of chemical peritonitis in a patient on peritoneal dialysis. *Clin Nephrol* 1995; **43**: 136–137.
3. Nolan DG. Inflammatory peritonitis with ascites after methylene blue dye chromopertubation during diagnostic laparoscopy. *J Am Assoc Gynecol Laparosc* 1995; **2**: 483–485.
4. Cornelis T, Bargman JM. Sudden increase in 'urine' output in a peritoneal dialysis patient. *Perit Dial Int* 2010; **30**: 574–576.

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The Authors Reply: We appreciate Dr Fourtounas[1] for his interest in our work.[2] We agree that the unexpected glycosuria and the abdominal computed tomography imaging highly suggested that the urinary bladder might be perforated by the peritoneal dialysis (PD) catheter. However, we think a confirmatory test is still needed to make the decision to remove a newly inserted PD catheter. The reason for Dr Fourtounas' objection to use methylene blue-stained dialysate in PD patients is fear of chemical peritonitis. We fully agree that nephrologists should try to preserve peritoneum as long as possible. However, we searched the literature on this issue and found only two case reports that are also mentioned in the letter.[1] In addition, we found seven PD patients who have been reported to receive intraperitoneal use of methylene blue-stained dialysate and video-assisted thoracoscopic surgery to detect and repair their pleuroperitoneal communications.[3–6] None of them developed chemical peritonitis and all of them resumed PD well. Therefore, whether the use of methylene blue-stained dialysate for diagnostic purposes is contraindicated in PD patients is still a matter of debate. Furthermore, in this patient, the immediate presence of blue urine in the urinary bag indicated that most of the methylene blue-stained dialysate was infused directly into the urinary bladder, rather than the peritoneal cavity.

1. Fourtounas C. Methylene blue and peritoneal dialysis. *Kidney Int* 2011; **79**: 136.
2. Wu HH, Li SY, Yang WC. Make your diagnosis: adynamic ileus after insertion of peritoneal dialysis catheter. *Kidney Int* 2010; **78**: 525–526.
3. Wang HB, Kao CC, Hsu KF *et al.* Diagphragmatic bleb complicated hydrothorax in peritoneal dialysis. *Inter Med* 2009; **48**: 1333–1334.
4. Tang S, Chui WH, Tang AW *et al.* Video-assisted thoracoscopic talc pleurodesis is effective for maintenance of peritoneal dialysis in acute hydrothorax complicating peritoneal dialysis. *Nephro Dial Transplant* 2003; **18**: 804–808.
5. Tsunezuka Y, Hatakeyama S, Iwase T *et al.* Video-assisted thoracoscopic treatment for pleuroperitoneal communication in peritoneal dialysis. *Eur J Cardiothorac Surg* 2001; **20**: 205–207.
6. Hosoda H, Nishio Y, Fujisaki H *et al.* Videoscopic surgical treatment for the patient of pleuroperitoneal communication complicating CAPD. *Kyobu Geka* 2000; **53**: 251–253.

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Effects of angiotensin-converting enzyme inhibitors and angiotensin II receptor blockers on peritoneal transport

To the Editor: I have read the excellent revision by Nessim *et al.*[1] entitled 'The renin–angiotensin–aldosterone system in peritoneal dialysis: is what is good for the kidney also good for the peritoneum?'. In the part of the article where the authors describe the potential strategies for peritoneal membrane preservation, they stated that the first study in this area with human data was published in 2007 by Kolesnyk *et al.* in *Peritoneal Dialysis International*.[2] However, this is incorrect, because the first study, published in *Nephron*, about the effects of angiotensin-converting enzyme inhibitors (ACEIs) on peritoneal membrane transport was a short-term study that included 12 diabetic patients on continuous ambulatory peritoneal dialysis and that showed the decrease of peritoneal protein losses with the oral administration of captopril, as

early as in 1989, by Coronel *et al.*[3] This effect was also demonstrated again by our group in 2004 in patients on peritoneal dialysis (PD) with an angiotensin receptor blocker (irbesartan); besides the decrease of peritoneal protein leakage and proteinuria, irbesartan was also shown to induce changes in other peritoneal membrane functions.[4] Both studies are mentioned by Kolesnyk *et al.*[5] in their recent review as the first studies in PD patients with this approach.

Studies with ACEIs, angiotensin receptor blockers, or other substances that can modify peritoneal transport and maybe improve technique survival still are needed, but we wish to point out our modest contribution.

1. Nessim SJ, Perl J, Bargman JM. The renin-angiotensin-aldosterone system in peritoneal dialysis: is what is good for the kidney also good for the peritoneum? *Kidney Int* 2010; **78**: 23–28.
2. Kolesnyk I, Dekker FW, Noordzij M *et al.* Impact of ACE inhibitors and AII receptor blockers on peritoneal membrane transport characteristics in long-term peritoneal dialysis patients. *Perit Dial Int* 2007; **27**: 446–453.
3. Coronel F, Hortal L, Naranjo P *et al.* Captopril, proteinuria and peritoneal protein leakage in diabetic patients. *Nephron* 1989; **51**: 443.
4. Coronel F, Berni A, Cigarrán S *et al.* Effect of angiotensin II receptor blocker (irbesartan) on peritoneal membrane functions. *Adv Perit Dial* 2004; **20**: 27–30.
5. Kolesnyk I, Struijk DG, Dekker FW *et al.* Effects of angiotensin-converting enzyme inhibitors and angiotensin II receptor blockers in patients with chronic kidney disease. *Netherlands J Med* 2010; **68**: 15–22.

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The Authors Reply: We thank Dr Coronel for his comments.[1] Indeed, over two decades ago, Coronel *et al.*[2] examined the effects of blockade of the renin–angiotensin–aldosterone system (RAAS) on peritoneal membrane function. Although the two studies mentioned were both small with a short exposure time to RAAS blocking agents,[2,3] they were the earliest studies to report the effect of blocking the RAAS on peritoneal membrane function in peritoneal dialysis patients, setting the stage for the subsequent, larger-scale observational studies by Kolesnyk *et al.*[4,5] Unfortunately, we were limited in the number of references for this mini-review, and did not have the opportunity to discuss these smaller early studies.

1. Coronel F. Effects of angiotensin-converting enzyme inhibitors and angiotensin II receptor blockers on peritoneal transport. *Kidney Int* 2011; **79**: 136–137.
2. Coronel F, Hortal L, Naranjo P *et al.* Captopril, proteinuria and peritoneal protein leakage in diabetic patients. *Nephron* 1989; **51**: 443.
3. Coronel F, Berni A, Cigarran S *et al.* Effects of angiotensin II receptor blocker (irbesartan) on peritoneal membrane functions. *Adv Perit Dial* 2004; **20**: 27–30.
4. Kolesnyk I, Dekker FW, Noordzij M *et al.* Impact of ACE inhibitors and AII receptor blockers on peritoneal membrane transport characteristics in long-term peritoneal dialysis patients. *Perit Dial Int* 2007; **27**: 446–453.
5. Kolesnyk I, Noordzij M, Dekker FW *et al.* A positive effect of AII inhibitors on peritoneal membrane function in long-term PD patients. *Nephrol Dial Transplant* 2009; **24**: 272–277.

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The missing 'interstitial vasculitis'

To the Editor: In a recent review on acute interstitial nephritis (AIN),[1] the authors state that AIN may be associated with systemic disorders such as sarcoidosis, Sjogren, or systemic lupus erythematosus, but they did not mention the 'interstitial vasculitis', that is, AIN as the only expression of renal vasculitis.

However, the occurrence of an involvement confined to the capillaries of renal tubular and interstitial structures in the setting of systemic vasculitides has been clearly stated in previous papers published by the European Vasculitis Study group[2] and others.[3]

Although interstitial vasculitis associated with glomerular vasculitis is a common observation, interstitial vasculitis as the only expression of renal involvement in systemic vasculitides is a rare entity reported in 1% of cases. Pathogenic mechanisms are presumed to be the same as described in systemic vasculitis, eventually leading to the destruction of the capillary walls of peritubular and interstitial capillaries due to the consequences of inflammatory reaction from the infiltrated cells.

However, the most intriguing point is the unknown reasons for which the occurrence of this kind of vasculitis only confined to the tubular and interstitial capillary walls is so rare an event. Some reports of the literature[3,4] and our personal experience in one case might suggest the existence of a specific nosographic entity characterized by the co-existence of giant cell (temporal) arteritis, polymyalgia rheumatica, and renal interstitial vasculitis. Therefore, we

Table 1 | Etiology of biopsy-proven acute interstitial nephritis (modified from Praga and González[1])

Drugs	Antibiotics, NSAIDs, acyclovir, allopurinol, furosemide, omeprazole, etc.
Infections	Bacteria, viruses, others
Associated with systemic diseases	Sarcoidosis, Sjogren, systemic lupus erythematosus, vasculitis
Idiopathic	Anti-TBM, etc.